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C H A P T E R 5

Follow-up of children born with an umbilical arterial blood pH < 7

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Abstract**Objective**

We performed neurodevelopmental assessment in children born with an umbilical artery pH < 7.

Methods

All infants born with an umbilical artery pH < 7 from a 19-month period were retrieved from the obstetric database. Obstetric, neonatal, and pediatric records were reviewed. At an age of 1 to 3 years, children were visited at home for semi-structured questioning of the mother and a Denver Developmental Screening Test of the child.

Results

During the study period 1614 umbilical artery pH measurements were entered in the database. Thirty (1.9%) were < 7. From this group 23 infants were admitted to the neonatal intensive care unit, and 8 of them required intubation. Twenty-eight children survived the neonatal period. Three children experienced an episode of mild hypertonia. One child had a mild motor developmental delay.

Conclusion

Babies born with an umbilical artery pH < 7 are at great risk to experience considerable short-term morbidity. Those who leave the neonatal intensive care unit without major problems have good outcomes, and pessimism in counselling their parents is unwarranted.

Introduction

Umbilical artery pH is considered to be an objective measurement, reflecting the baby's condition at the time of birth. In many countries this measurement is part of quality control programs in obstetric care and may be used in cases of litigation. This pH value correlates well with fetal scalp blood pH immediately before delivery and with capillary blood pH of the baby immediately after birth.¹ During pregnancy umbilical artery pH averages 7.37 (SD 0.03). Published normal and pathologic values after delivery are listed in **Table 1**.¹⁻¹⁵ The lower statistical limits of normal umbilical artery pH values (mean - 2 SD) in this table range from 7.02 to 7.18. An umbilical artery pH < 7 is well below this range and has been suggested before to be the most realistic cutoff for pathologic acidemia at birth.^{16,17}

Table 1. Umbilical artery pH values reported in literature

| Source | No. of deliveries | Mean umbilical artery pH | Lower statistical limit of normal umbilical artery pH (mean - 2 SD) |
|---|-------------------|--------------------------|---|
| Saling ¹ (1964) | 77* | 7.25 | 7.09 |
| Kubli <i>et al.</i> ² (1972) | 3,317 | -† | < 7.10 (5%) |
| Römer <i>et al.</i> ³ (1976) | 3,804 | 7.27 | 7.10 |
| Huisjes and Aarnoudse ⁴ (1979) | 852 | 7.20 | 7.02 |
| Sykes <i>et al.</i> ⁵ (1982) | 899 | 7.20 | 7.04 |
| Eskes <i>et al.</i> ⁶ (1983) | 4,667 | 7.23 | 7.09 |
| Yeomans <i>et al.</i> ⁷ (1985) | 146* | 7.28 | 7.18 |
| Low ⁸ (1988) | 4,500 | 7.26 | 7.13 |
| Ruth and Raivio ⁹ (1988) | 106* | 7.29 | 7.15 |
| Ramin <i>et al.</i> ¹⁰ (1989) | 1,292* | 7.28 | 7.14 |
| Thorp <i>et al.</i> ¹¹ (1989) | 1,694* | 7.24 | 7.10 |
| Fee <i>et al.</i> ¹² (1990) | 13,601 | 7.27 | -† |
| Miller <i>et al.</i> ¹³ (1990) | 147* | 7.27 | 7.15 |
| Römer and Wesseler ¹⁴ (1991) | 2,549 | 7.27 | 7.13 |
| Vintzileos <i>et al.</i> ¹⁵ (1992) | 243* | 7.28 | 7.14 |
| This study | 1,614 | 7.21 | 7.03 |

*Selected population (uncomplicated pregnancy and delivery).

†Values not reported by authors.

Low umbilical artery pH values tend to be viewed with a pessimism similar to that for low Apgar scores. Several authors have pointed out that pediatricians and obstetricians tend to be unrealistically pessimistic about the prognosis of infants born with low Apgar scores.^{18,19} The same applies to low umbilical artery pH values, although umbilical artery acidemia at birth is seldom associated with poor neurologic outcome.^{9,12,20} Dijkhoorn *et al.* concluded that most neonatal neurologic abnormalities must be caused by other factors.²¹ Definitions of acidemia, however, differ among studies, and follow-up is often rather selective. Realistic counselling of parents as to what to expect in the long run is therefore difficult. To provide a rational basis for such counselling we conducted a follow-up of all infants born in our unit with an umbilical artery pH < 7 and studied their developmental outcome at age 1 to 3 years.

Methods

Routine measurement of both venous and arterial umbilical pH was introduced for all births at Leiden University Hospital in April 1991. A segment of the cord 10 to 30 cm long is doubly clamped and kept at room temperature. Blood gas and pH are measured within 30 minutes after birth, with a Corning 178 (Medfield, Mass.) analyzer. This analyzer, located next to the delivery rooms, is tested twice a day to verify its reliability.

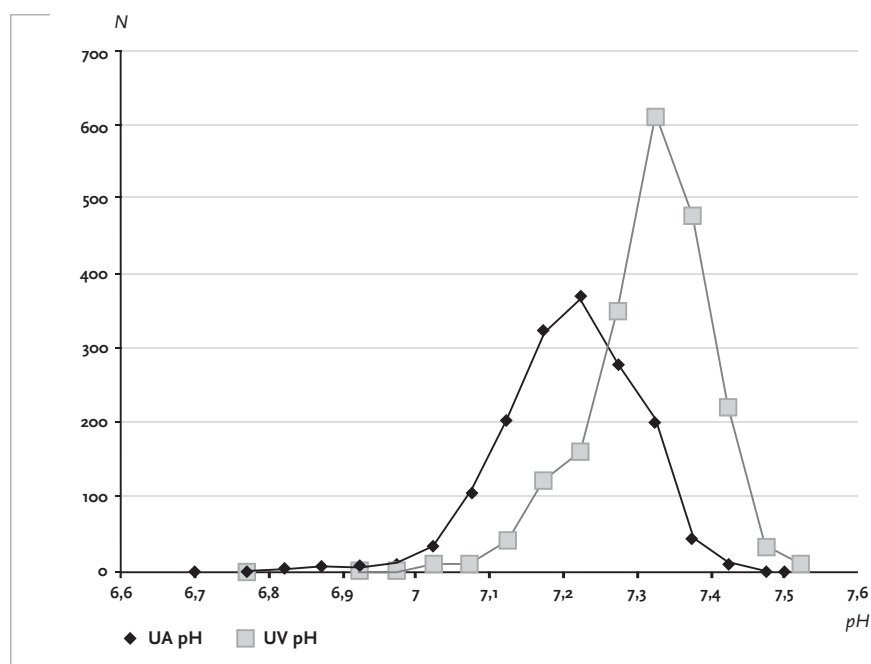
Data were entered in our obstetric database. The database was searched for umbilical artery pH values < 7 of all babies born alive, of at least 24 weeks' gestation, and without apparent congenital anomalies for the period up to December 1992. Obstetric and neonatal records of these mothers and babies were reviewed.

With approval of the protocol by the hospital's ethical committee, family physicians were contacted by letter to explain the aims and nature of the study. After consultation with the family physician, the parents were sent an explanatory letter asking their permission to review all medical records and their cooperation for screening the children in their home environment. At the subsequent home visit a history was taken from the mother, concentrating on her birth experience and development of the child. The child was examined with the Dutch version of the Denver Developmental Screening Test (DDST). The DDST is designed for children aged 2 weeks to 6 years and covers four areas of development: personal social, fine motor adaptive, language, and gross motor development. The test results are scored as normal, questionable, or abnormal. All home visits were performed in January and February of 1994 by one of the authors (HTCN) who had received 3 months' intensive training in pediatric neurology and neuropsychology for this purpose.

Results

During the 19-month study period 2,536 babies were born in the department. Umbilical vein and umbilical artery pH, respectively, were available for 81% and 64% of them. Mean umbilical vein pH was 7.31 (SD 0.09), and mean umbilical artery pH was 7.21 (SD 0.09) (**Fig 1**). Mean arteriovenous difference was 0.11 (SD 0.06).

Figure 1. Frequency distribution of umbilical vein (UV) and umbilical artery (UA) pH values measured between April 1991 and December 1992 at the Obstetric Department of Leiden University Hospital.



Thirty (1.9%) of the 1,614 babies with recorded umbilical artery pH values had a value < 7. Umbilical artery pH at birth and 5-minute Apgar score of these babies showed only a weak correlation ($r = 0.45$) (**Fig. 2**). The obstetric data of the 30 babies are summarized in **Table 2**. Mean umbilical artery blood pH in this group was 6.91 (range 6.70 to 6.99). Mean umbilical artery base deficit in this group was 16.85 (range 11.4 to 26.5). Nine of the 30 babies (30%) were preterm (< 37 weeks), but only one of them was very preterm (< 32 weeks) whereas 6 (20%) were postterm (**Table 2**). All neonates except one (case 15) with a birth weight of 2,380 g at 38 weeks were of appropriate weight for gestation. In 20 of the 30 infants fetal distress was suspected on the basis of an abnormal fetal heart

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rate pattern or fetal scalp acidemia during delivery. Two babies were born after emergency cesarean section because of complications during cordocentesis for suspected fetal thrombocytopenia. In both cases a cord hematoma was seen after birth. In one (case 10, maternal idiopathic thrombocytopenia) the fetal platelet count was $164,000/\mu\text{l}$. In the other (case 14, alloimmune thrombocytopenia on the basis of human platelet antigen-1a) the fetal platelet count during the procedure was $32,000/\mu\text{l}$.

Figure 2. Correlation between umbilical artery (UA) pH at birth and 5-minute (5') Apgar score in 30 infants born with an umbilical artery pH < 7. Umbilical vein (UV) pH was < 7 in 7 cases.

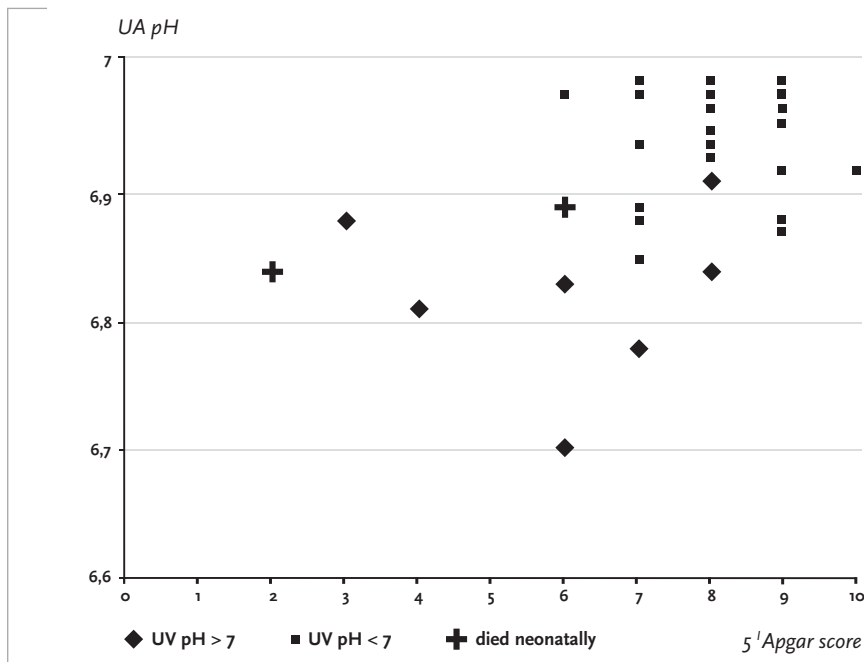


Table 2 (next page) also shows neonatal and pediatric data of these 30 children.²² Of the 23 infants admitted to the neonatal intensive care unit, 8 required intubation and artificial ventilation. Because of clear signs of severe neurologic damage, artificial ventilation was discontinued in 2 neonates. They both died shortly thereafter. Of the 28 survivors, 25 could be visited at home. For 3 of the infants the parents gave permission to retrieve all medical information but declined a home visit. Reasons for refusal were excessive medical contacts (case 7), feelings of resentment (case 12), and not given (case 18). The medical records of all 3 explicitly mentioned normal neurologic development at the ages of 14 (case 7), 26 (case 12), and 29 (case 18) months, respectively.

The ages of the children at the home visits ranged from 14 to 33 months. Twenty-three infants performed the DDST well and scored a normal test result. One child (case 24) displayed a mild motor developmental delay. Her test result was scored as questionable. Another child (case 9) refused to perform some items of the fine motor adaptive tasks. Her test result was scored as questionable, but she was tested shortly afterward by a pediatrician and then performed the test in a normal manner. None of the children had results scored as abnormal.

Three children had experienced an episode of mild hypertonia. Febrile convulsions had occurred in 3 children, in two of them more than once. In both cases neurologic examination and electroencephalographic findings showed no abnormalities.

The semi structured interview of the 25 mothers visited at home resulted in the following assessments. Twenty-one were aware of the fact that an Apgar score was given, and 19 could explain the main principles of the score. Only 4 knew, before they received our request to participate in this study, that umbilical artery pH measurement was routinely performed. Nineteen of 25 mothers believed that their babies did not have a good start in life.

Discussion

We studied a complete cohort of children with severe acidemia at birth defined as an umbilical artery pH < 7. There was considerable short-term morbidity: 77% were admitted to the neonatal intensive care unit and 27% required artificial ventilation. Two of the 30 children died in the neonatal period. In a follow-up of the 98 survivors we found no major abnormalities at age 1 to 3 years. During their first years of life, 4 of the 28 (14%) surviving children demonstrated minor abnormalities: three had episodes of mild hypertonia, and one had a mild motor developmental delay as detected by the DDST. This incidence is comparable to the 21% incidence of mild motor abnormalities found by Low *et al.* in the first year after uneventful birth.²³ We do realize, however, that the DDST is only a screening test and that subtle forms of brain damage may not be discovered by this test.

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Table 2. Obstetric, neonatal, and pediatric data

| Case No. | Obstetric | | | | | | Neonatal | | |
|----------|--------------------------------|-------|-------|--|-----------------------------|---|-----------------|---------------------------|--|
| | Gestational age (compl. weeks) | UA pH | UV pH | Obstetric risk factors | Suspicion of fetal distress | Mode of delivery | NICU Admittance | Length of intubation (hr) | Clinical signs of encephalopathy ²³ |
| 1 | 32 | 6.88 | 6.94 | Preeclampsia, abruption placentae | + | CS before labor | + | 72 | - |
| 2 | 33 | 6.91 | 6.95 | Uterine rupture | + | CS during labor | + | 6 | - |
| 3 | 34 | 6.89 | 7.03 | Diabetes mellitus | + | CS before labor | + | - | - |
| 4 | 35 | 6.78 | 6.80 | Abruptio placentae | + | CS before labor | + | 1 | Sarnat I |
| 5 | 36 | 6.70 | 6.75 | Abruptio placentae | + | CS before labor | + | - | - |
| 6 | 36 | 6.88 | 7.31 | - | - | Spontaneous | - | - | - |
| 7 | 36 | 6.92 | 7.23 | - | - | Spontaneous | - | - | - |
| 8 | 36 | 6.98 | 7.37 | PROM; breech | + | Extraction | + | - | - |
| 9 | 36 | 6.99 | 7.28 | Rh isommunization; Breech | + | Extraction | + | - | - |
| 10 | 37 | 6.92 | 7.08 | Complication at cordocentesis | + | CS before labor | + | 3 | - |
| 11 | 37 | 6.99 | 7.20 | Breech | - | Extraction | + | - | - |
| 12 | 38 | 6.81 | 6.94 | PROM | - | Vacuum extraction | + | - | Sarnat I |
| 13 | 38 | 6.84 | - | Abruptio placentae | + | CS before labor | + | 100 | Sarnat III |
| 14 | 38 | 6.89 | 7.31 | Complication at cordocentesis | + | CS before labor | + | 14 | Sarnat III |
| 15 | 38 | 6.99 | 7.04 | PIH | + | Trial of vacuum extraction; CS during labor | + | 3 | - |
| 16 | 39 | 6.84 | 6.90 | Meconium | - | Spontaneous | + | - | - |
| 17 | 39 | 6.94 | 7.23 | Breech | + | Extraction | + | - | - |
| 18 | 39 | 6.96 | 7.01 | HELLP | - | Spontaneous | - | - | - |
| 19 | 39 | 6.98 | 7.14 | - | + | Spontaneous | - | - | - |
| 20 | 40 | 6.85 | 7.17 | Intrapartum version of oblique lie | + | CS during labor | + | 70 | - |
| 21 | 40 | 6.94 | 7.04 | PROM; meconium | + | Vacuum extraction | + | - | - |
| 22 | 41 | 6.83 | 6.99 | No progress | + | Vacuum extraction | + | - | Sarnat I |
| 23 | 41 | 6.99 | 7.10 | CPD; meconium | + | CS during labor | + | - | - |
| 24 | 41 | 6.99 | 7.28 | - | - | Vacuum extraction | - | - | - |
| 25 | 42 | 6.89 | 7.02 | Epileptic convulsions during pregnancy | - | Vacuum extraction | + | - | - |
| 26 | 42 | 6.89 | 7.14 | Breech | + | Extraction | + | - | Sarnat I |
| 27 | 42 | 6.94 | 7.06 | Breech; meconium; cord prolapse | + | CS during labor | + | - | Sarnat I |
| 28 | 42 | 6.95 | 7.29 | No progress | - | Vacuum extraction | - | - | - |
| 29 | 42 | 6.97 | 7.27 | Meconium | + | Spontaneous | - | - | - |
| 30 | 42 | 6.99 | 7.08 | - | - | Spontaneous | + | - | - |

78 UA, umbilical artery; UV, umbilical vein; NICU, neonatal intensive care unit; plus sign, present; minus sign, absent; CS, cesarean section; N, normal result; PROM, premature rupture of membranes;

| signs of renalopathy | Neonatal | | | | Pediatric | | |
|-------------------------|--|------------------------------------|-------------------------|---|---|------------------------|------|
| | Pulmonary problems | Renal or liver dys- function | Antibiotic Treatment | Other neonatal complications | Medical history | Age at test (mo) | DDST |
| | Hyaline membrane disease, grade III | - | + | Bradycardia | Febrile convulsions; systolic murmur | 31 | N |
| | - | - | + | Hypoglycemia | - | 25 | N |
| | - | - | - | Myocardial hypertrophy | Delayed bonding | 17 | N |
| | Wet lung | + | - | Leukopenia; hypoglycemia | Pyloromyotomy | 19 | N |
| | - | + | - | - | Hypertonia; febrile convulsions | 28 | N |
| | - | - | - | - | - | 31 | N |
| | - | - | - | - | Inguinal hernia; amblyopia | - | - |
| | - | - | - | Jaundice | - | 21 | N |
| | - | - | - | Exchange transfusion | Myocardial hypertrophy | 24 | Q |
| | Wet lung | - | - | - | - | 32 | N |
| | - | - | - | Jaundice | - | 32 | N |
| | - | - | + | - | - | - | - |
| | Pneumothorax | + | + | Hypovolemic shock; convulsions; died 100 hr postpartum | | | |
| | - | + | + | Generalized hypotonia; died 18 hr postpartum | | | |
| | - | - | - | Jaundice | Hypertonia | 26 | N |
| | Wet lung | - | - | - | Otitis media; pulled elbow | 33 | N |
| | - | + | - | Brain scan; frontal flaring | Normal brain scan | 21 | N |
| | - | - | - | - | Single febrile convulsion | - | N |
| | - | - | - | - | Hyperactive | 28 | N |
| | Meconium aspiration | - | + | - | - | 25 | N |
| | - | + | + | Sepsis; meningitis | - | 24 | N |
| | - | + | - | - | Bronchial asthma | 19 | N |
| | - | - | - | Single umbilical artery | Atopy; hypertonia | 17 | N |
| | - | - | - | - | Obstipation | 14 | Q |
| | - | - | - | Jaundice; hypoglycemia | - | 24 | N |
| | - | - | + | - | - | 29 | N |
| | - | - | - | Hypertonia | Sacral dimple | 16 | N |
| | - | - | - | - | - | 14 | N |
| | - | - | - | - | - | 30 | N |
| | - | - | - | - | - | 18 | N |

Q, questionable result; PIH, pregnancy induced hypertension; HELLP, hemolysis, elevated liver enzymes, low platelet count; CPD, cephalopelvic disproportion.

We searched the literature for follow-up studies on children born with severe acidemia. In the studies by Ruth and Raivio and Dijkhoorn *et al.* no major motor or cognitive deficits were found among children born with umbilical artery pH < 7.^{9,24} The number of infants with pH < 7 studied in these reports was extremely small and follow-up ranged from 1 to 12 months. Fee *et al.* studied 15 children and Dennis *et al.* studied 27 children born with an umbilical artery pH < 7.05.^{12,20} These children were followed up at age 1 to 2 years and age 4 to 5 years, respectively. None of these children were reported to have major deficits. Goodwin *et al.*, however, reported major abnormalities among 10 of 29 infants born with an umbilical artery pH < 7.²⁵ In their study umbilical artery pH measurement was selectively done in cases of fetal distress or neonatal depression. Follow-up was restricted to those infants with abnormal examination results at hospital discharge. There was no follow-up of infants who were normal at discharge from the neonatal intensive care unit.

Low *et al.* conducted two follow-up studies on children born with metabolic acidosis, defined as buffer base < 34 mEq/L.^{23,26} In a first study 37 infants without neonatal encephalopathy were selected.²³ At follow-up one child had a major motor handicap caused by a traumatic intercurrent event during childhood. They found no other children with major motor or cognitive deficit in the study group. In a second study the same authors evaluated another 37 infants.²⁶ Five of these had major motor deficits at age 1 year and 2 of these 5 infants also were mentally retarded. In contrast to the findings in the former study, almost half of the infants included in this study had documented newborn encephalopathy. Three of the 5 infants with major deficits had severe newborn encephalopathy with coma or multiple seizures.

Conclusion

Umbilical artery pH measurement is not superfluous. It has actual value in selecting those babies that are in need of extra neonatal care, and it also provides good means of retrospectively evaluating our obstetric efforts in preserving fetal health during birth. The results of our study indicate, however, that umbilical artery pH measurement after birth is not predictive of serious developmental delay, unless it is accompanied by clinical evidence of hypoxic encephalopathy. This seems only logical, since this measurement remains no more than a snapshot of the situation. In addition to the degree of acidosis, the duration of acidemia undoubtedly plays a role. To summarize our results we would state that if a neonate born with severe acidemia shows no severe neurologic abnormalities in the newborn period, pessimism in counselling the parents concerning the future psychomotor development of their child is unwarranted.

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